Supplementary Table 1. Overview of case reports with acquired neurogenic stuttering in the literature cohort

#	Paper	Age	Sex	Hand.	Location	Side	Delay	Other speech disorders	Other symptoms	Lesion type
1	Ardila 1986	50	M	R	Temporal lobe	R	"After the recovery"	Speaking difficulty	L hemiparesis	Ischaemic stroke
2	Soroker 1990	65	M	R	Internal capsula, putamen, periventricular white matter	R	2 weeks	-	L hemiparesis	Ischaemic stroke
3	Abe 1993	38	M	-	Thalamus, midbrain	Bil	< 3 months	-	Tetraparesis, apathy, gaze palsy	Ischaemic stroke
4	Grant 1999 Case 1	68	M	R	Frontotemporoparietal region	L	No delay	Dysarthria	R hemiparesis	Ischaemic stroke
5	Grant 1999 Case 4	55	M	R	Occipital lobe	L	< 2 weeks	-	R hemianopia	Ischaemic stroke
6	Carluer 2000	58	M	R	Striatum	L	Weeks	Transient aphasia	R hemiparesis and parkinsonism	Ischaemic stroke
7	Ciabarra 2000 Case 1	53	M	R	Rostromedial pons	L	No delay	Dysarthria	Vertigo, ataxia, L INO, L facial droop, jaw tremor	Ischaemic stroke
8	Ciabarra 2000 Case 2	54	F	R	Putamen, caudate, corona radiata	L	No delay	Dysarthria	R facial droop, slowness of right finger movements	Ischaemic stroke
9	Ciabarra 2000 Case 3	63	F	L	Corona radiata, putamen, subinsular	L	No delay	Difficulty finding words	Reduced vision, R hemiparesis	Ischaemic stroke
10	Turgut 2002	61	M	R	Parietal cortex	L	No delay	-	R hemiparesis	Ischaemic stroke
11	Doi 2003	60	M	-	Midbrain, pons	Bil	No delay	Dysarthria	Vertigo, ataxia, gaze palsy	Ischaemic stroke
12	Van Borsel 2003	38	M	R	Thalamus	L	< 6 months	Aphasia	R hemiparesis	Ischaemic stroke
13	Kakishita 2004	51	M	R	Corpus callosum	R	No delay	-	-	Ischaemic stroke
14	Hamano 2005	77	F	R	Corpus callosum	Mid	No delay	-	Dizziness, nausea	Ischaemic stroke
15	Sahin 2005 Case 1	65	F	R	Parietal lobe	L	2 days	Transient disability to speak	R hemiparesis	Ischaemic stroke
16	Osawa 2006	51	M	R	Tempo-parieto-occipital	L	< 2 days	Wernicke's aphasia	Homonymous hemianopsia, apraxia	Ischaemic stroke
17	Karakis 2008	51	F	R	Midbrain	Mid	1 week	Dysarthria	Gaze palsy, ataxia	Ischaemic stroke
18	Tani 2011 Case 1	49	M	A	Putamen	Bil	days	Anomic aphasia, dysathria	R hemiparesis	Haemorrhage
19	Tani 2011 Case 2	16	M	R	Putamen, globus pallidus	Bil	6 months	Dysarthria	Ataxia	Trauma
20	Van Houtte 2014	28	F	R	Temporal lobe	L	days	-	Transient linguistic disturbances	Haemorrhage

Lesion location is listed according to the description in the original case report.
A = ambidextrous, Bil = bilateral, F = female, Hand = Handedness, INO = internuclear ophtalmoplegia, L = left, M= male, Mid=Midline, R = right, - = Not reported.

Supplementary Table 2. Demographics of the cases in the acquired and developmental stuttering datasets

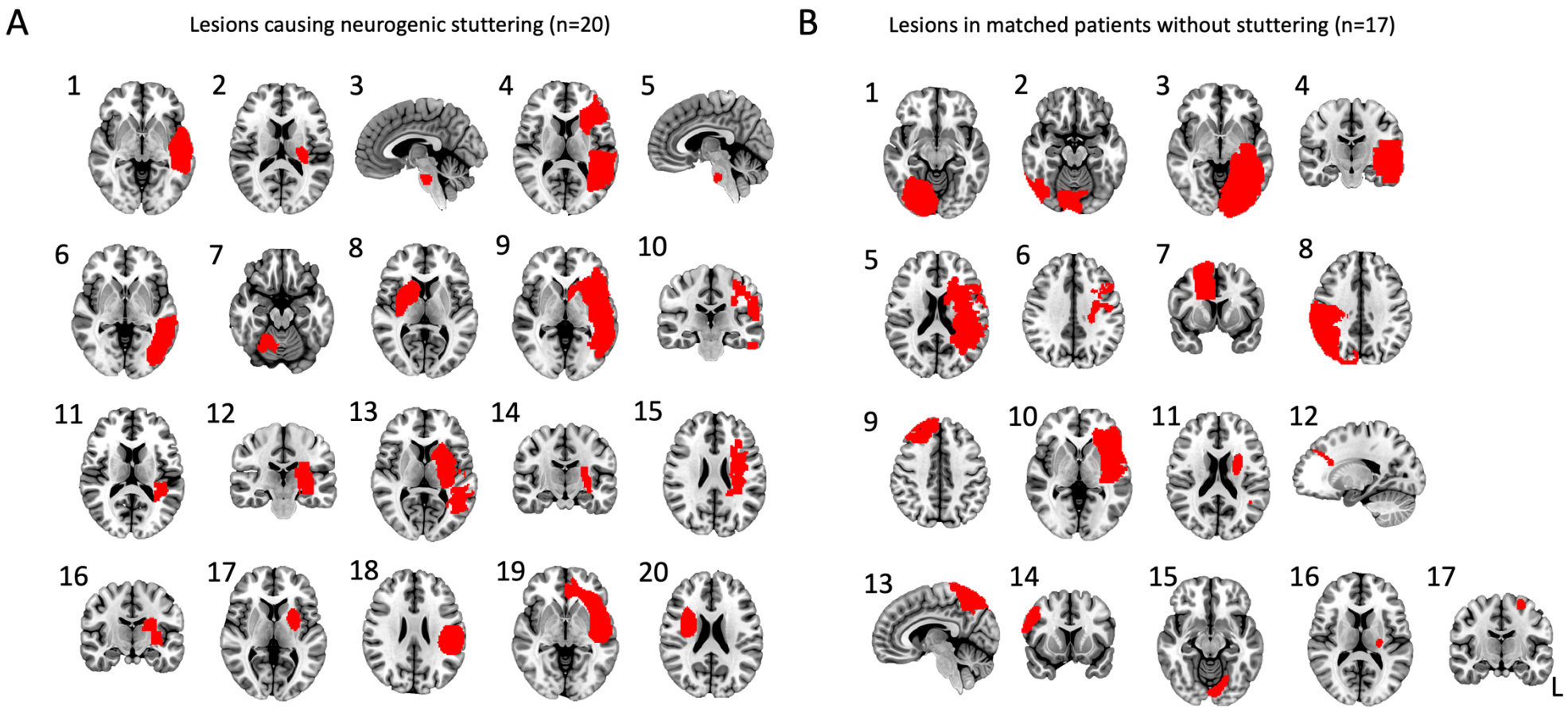
	Neurogenic stuttering – literature cohort	**Controls – literature cohort**	**Neurogenic stuttering - clinical cohort**	**Controls – clinical cohort**	**Developmental stuttering cohort**
N	20	169	20	17	20
Age (median [range])	53 [16-77]	69 [27-92]	72 [45-87]	69 [50-83]	53 [18-43]
Sex (M/F)	14/6	105/64	13/7	11/6	14/6
Handedness	13R, 1A, 6L	106R, 6L[1]	20R	17R, 3L	15R, 4A, 1L
Aetiology (n [%])	17 [85%] infarcts, 3 [15%] haemorrhages	139 [87%] infarcts, 16 [10%] haemorrhages, 5 [3%] both[2]	18 [90%] infarcts, 2 [10%] haemorrhages	15 [88%] infarcts, 1 [12%] haemorrhage	N/A
% stuttered disfluencies during conversation (median [range])[3]			4.5 [1.8-19.4][4]	1.5 [0.3-2.6]	4.5 [0.9-35.2]
% stuttered disfluencies during monologue (median [range])			4.4 [1.8-10.9][5]	1.0 [0.0-2.7]	
% stuttered disfluencies during reading (median [range])			3.0 [0.0-13.0][6]	0.5 [0.0-2.3]	
OASES score (median [range]) Impact (median [range])					2.5 [1.5-3.5] moderate [mild/moderate – moderate/severe]
Aphasia	4/11 (36%)		14/20 (70%)	10/15[4] (67%)	
Anomia	1/11 (9%)		13/20 (65%)	9/17 (53%)	
Dysarthria	7/11 (64%)		9/20 (45%)	4/17 (24%)	
Apraxia of speech			5/20 (25%)	2/17 (12%)	

[1]57 missing values
[2]9 missing values
[3]For the clinical cohort, interrater reliability was measured by rescoring of 25% of conversation samples (N = 10) by a trained second observer. The percentage of stuttered disfluencies was strongly correlated between the two raters (r_s=0.82, P<0.01).[34] For the developmental stuttering cohort, rescoring of 50% of conversation samples (N = 10) by a trained second speech therapist with expertise in stuttering showed a very strong correlation (r_s=0.99, P<0.01).
[4]The information from the clinical cohort has been published previously.[34] While the range of disfluencies across the various speech tasks may be broader, all participants were identified with acquired neurogenic stuttering if they presented with more than 3% stuttered disfluencies during at least one of the included speech tasks.
[5]7 missing values
[6]3 missing values
[7]2 missing values
R = right, A = ambidextrous, L = left, OASES = Overall Assessment of the Speaker's Experience of Stuttering[35]

Supplementary Table 3. Clusters showing significant associations with lesions in the literature cohort

Cluster Index	Voxels (N)	P Value	COG X (mm)	COG Y (mm)	COG Z (mm)	COG region
Positive Associations						
1	790	<.01	-29	0.06	-7.19	L putamen
2	108	0.01	30.5	-5.1	-10.9	R putamen
3	89	0.01	-38.5	21.8	11.7	L inferior frontal gyrus
Negative Associations						
1	1223	0.02	27.4	-75.1	43.6	R lateral occipital cortex
2	332	0.04	-23.5	-75.9	47	L lateral occipital cortex
3	21	0.05	57.7	-59.6	-16.2	L Inferior temporal gyrus
4	14	0.03	35	-49	67.9	R superior parietal lobule

COG = centre of gravity, L = left, R = right



Supplementary Fig. 1 Lesion maps of the clinical cohort. (A) Lesions causing acquired neurogenic stuttering (N = 20), **(B)** Lesions of the control group matched for occurrence of speech-language problems following stroke (N = 17). See Supplementary Table 2 for additional information.

Supplementary Table 4. Clusters showing significant associations with lesions in the clinical cohort

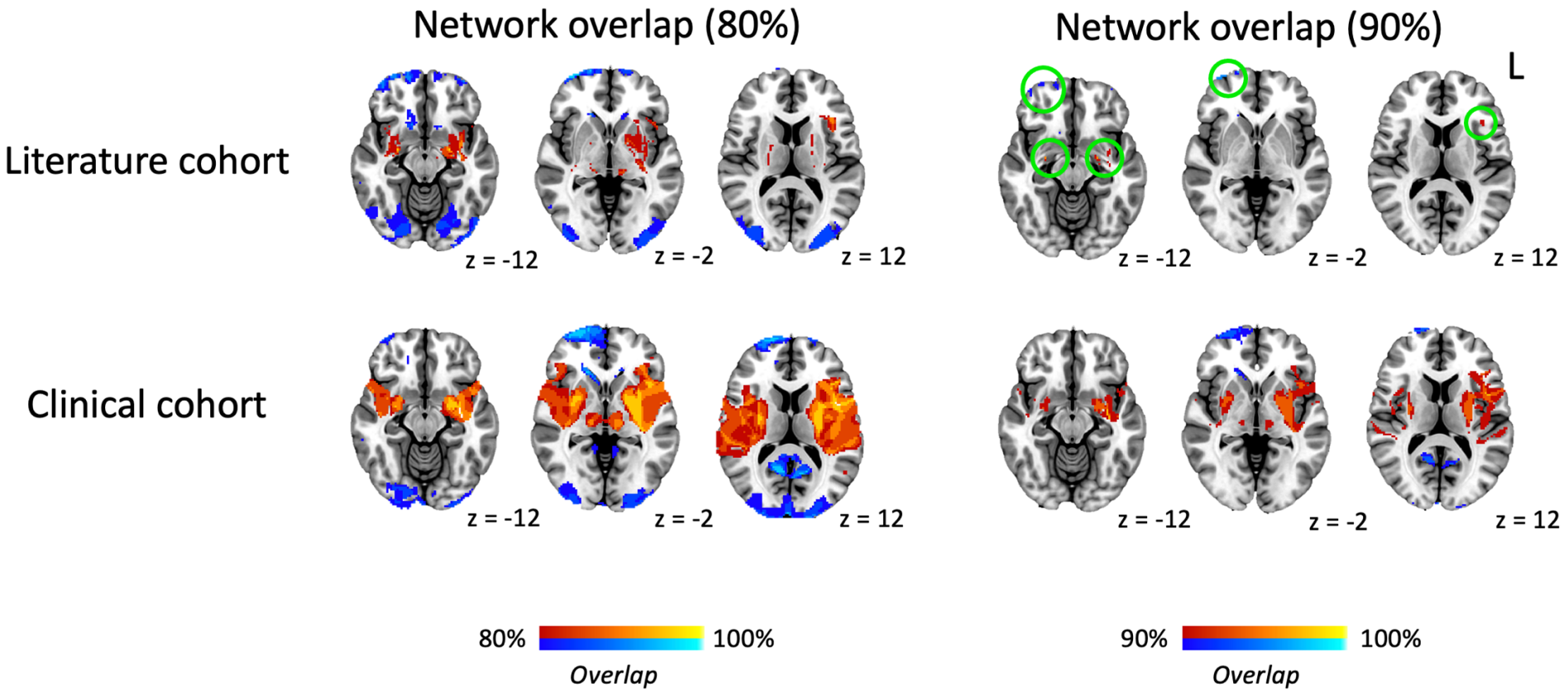
Cluster Index	Voxels (N)	P Value	COG X (mm)	COG Y (mm)	COG Z (mm)	COG region
Positive Associations						
1	1247	0.02	-31.3	-7.17	-6.51	L putamen
2	114	0.03	31.9	-0.69	-15.6	R amygdala
3	90	0.02	-6.4	-19.5	-36.6	L brainstem
4	69	0.04	39.7	-22.9	2.95	R Heschl's gyrus
5	53	0.01	-26.4	-1.24	-36.9	L parahippocampal gyrus/temporal fusiform cortex
6	32	0.03	-11.4	-21.5	-1.45	L thalamus
7	15	0.04	-14	-58.8	-24	L cerebellum

COG = centre of gravity, L = left, R = right

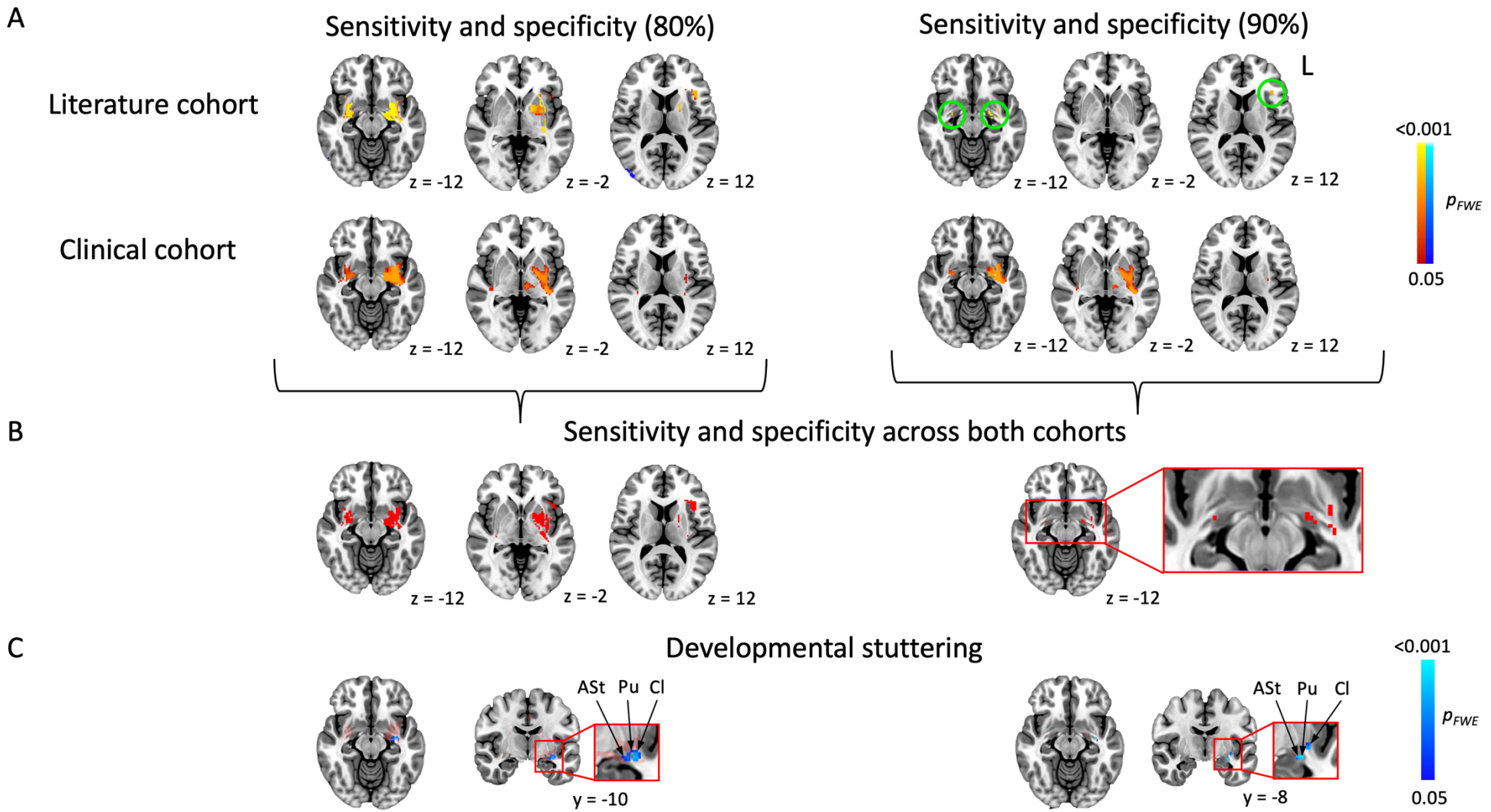
Supplementary Table 5. Common acquired stuttering hub, with clusters representing overlap of specificity data across the literature and clinical cohorts

Cluster Index	Voxels (N)	P Value	COG X (mm)	COG Y (mm)	COG Z (mm)	COG region
1	405	<0.01	-30	-5.15	-7.78	L putamen
2	22	<0.01	28.2	-4.18	-11.9	R amygdala

COG = centre of gravity, L = left, R = right



Supplementary Fig. 2 Lesion network overlap at different thresholds. Lesion network maps of the 20 cases were overlaid for each cohort, and thresholded at ≥80% overlap (left) and ≥90% overlap (right) to show regions connected to most of the lesion locations (i.e., regions sensitive to stuttering in the literature and clinical cohorts). Green circles used to highlight small clusters.



Supplementary Fig. 3 Confirmatory analyses with different lesion network overlap thresholds (80% on left, 90% on right). (A) Specificity analyses in the literature and clinical cohorts (whole brain P_{FWE}<0.05), followed by conjunction analyses using the 80% or 90% threshold for both groups (see Supplementary Fig. 2) to show areas both sensitive and specific for stuttering in each of the cohorts. Positive associations are shown in red-yellow, negative in blue-light blue. Green circles used to highlight small clusters. **(B)** Common acquired stuttering networks using the 80% (left) or 90% (right) threshold, showing common areas that were sensitive and specific across both neurogenic stuttering cohorts. **(C)** Regression analyses within the identified common acquired neurogenic stuttering networks (from B, shown as transparent red in C) showed that more negative experiences with stuttering (OASES scores) were associated with increased grey matter volume in participants with persistent developmental stuttering (P_{FWE}<0.05, shown in blue). ASt = amygdalostriatal transition area; Cl = claustrum; Pu = putamen.